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Sanocrysin Treatment.—Four hundred and thirty-eight patients with pulmonary tuberculosis were treated with sanocrysin. Of this number, 384 patients received sanocrysin as the only treatment. Tubercle bacilli disappeared from the sputum in 64 per cent of these. With ordinary sanatorium treatment, the percentage was 33, and with ordinary treatment and artificial pneumothorax it was fifty-six. The first group of 152 patients was treated chiefly with large dosages, and the second group of 232 with small doses. The results were decidedly more favorable in the second group, as the percentage in which bacilli disappeared was 70 as against 55 in the second group. This may be due to the fact that more suitable cases were selected in the latter. Sanocrysin is more effective in the treatment of fresh noncavernous lesions than in other types. It is also recommended as an adjuvant in collapse-therapy, as it seems to inhibit new lesions. The complications are slight, such as transient albuminuria and exanthema, diarrhoea and chrysiasis. The last-named complication is the precipitation of the gold into the skin, causing a characteristic slate-gray discoloration. Of the 12 deaths which occurred in the groups treated with sanocrysin, two were certainly due to the effect of the drug. In one case there was a diffuse hepatitis and in the other a dermatitis. However, it is the impression of the author that sanocrysin causes a shortening of the sanatorium-treatment period and a rise in the number of persons "fit for work" following such treatment.—*Sanocrysin Treatment, V. Stub-Christensen, Tubercle, November, 1931, xiii, 49.*—(*M. E. P.*)

Sanocrysin Treatment of Pulmonary Tuberculosis.—A group of 80 cases of tuberculosis given sanocrysin treatment was compared with 80 control patients classified into mild, moderate and severe types. Both groups were treated in identical fashion aside from the use of the sanocrysin. Of the sanocrysin cases 65 improved, as against only 29 per cent of the control cases. Tubercle bacilli disappeared in 39 of the 78 sanocrysin cases, while of the controls only 7 of 70 showed similar effects. Likewise, pyrexia disappeared more rapidly in the sanocrysin cases, and the sedimentation reaction was favorably influenced more frequently. The best results were obtained in cases that had existed less than a year. In cachectic cases treatment was without benefit. Outspoken intestinal tuberculosis was an absolute contraindication, while laryngeal tuberculosis was regarded as a relative contraindication.—*Über Sanocrysinbehandlung der menschlichen Lungentuberkulose, F. E. Koch, Beitr. z. Klin. d. Tuberk., 1930, lxxiii, 751.*—(*H. J. C.*)

Treatment of Pulmonary Tuberculosis with *Lopion*.—As the result of experience with 23 cases of pulmonary tuberculosis treated with *Lopion*, in which the author saw persistently high febrile reactions, he does not recommend the use of the material. Comparative results with *Triphel* proved this to be less toxic. With *Lopion* special caution must be used in the presence of

laryngeal tuberculosis, since even small doses can occasion persistent reactions in this condition.—*Über die Behandlung der Lungentuberkulose mit Lopion, Thiele, Beitr. z. Klin. d. Tuberk., 1930, lxxiv, 629.*—(*H. J. C.*)

Carbon Therapy in Tuberculosis.—Intravenous injections of very finely divided carbon (0.25–0.5 cc. of a carbon suspension prepared by Merck) caused a qualitative change in the blood-picture of healthy individuals, with increase of lymphocytes and monocytes. The same changes were noted in tuberculosis patients. In those with a favorable clinical course the same blood-picture was found as in normal persons. The injections were given at intervals of 10 to 14 days. The favorable action of the carbon injections is considered as due to a stimulation of the reticuloendothelial system, with resulting lymphocytosis and monocytosis.—*Studien zur Kohletherapie. I. Mitt. Veränderungen im Blute bei Tuberkulösen, R. Gross and J. Wadel, Beitr. z. Klin. d. Tuberk., 1930, lxxiv, 613.*—(*H. J. C.*)

Calcium and Tuberculosis.—The author verifies the usual findings of the constancy of the serum-calcium mirror, with an average value of between 10 and 11 mgm. per cent. In favorable productive processes the values lay above this while in severe forms of tuberculosis they were below normal. Asthenic cases without tuberculosis also showed a mild diminution. In tuberculosis the decrease paralleled a poor sedimentation reaction. Calcium Sandoz (the gluconic-acid compound of calcium) showed a favorable increase during treatment. Increases occurred after oral and intravenous injections, and persisted longest after intramuscular injection. Calcium treatment caused a diminution of sputum in numerous cases, night-sweats were favorably influenced, cough diminished, and the general well-being improved. In haemoptyses intravenous injections were preferable to other routes. Harmful effects were never noted.—*Calcium und Tuberkulose, J. Hein, Beitr. z. Klin. d. Tuberk., 1930, lxxiii, 569.*—(*H. J. C.*)

Treatment of Pulmonary Tuberculosis with Dead Tubercle Bacilli.—The author believes that the possibility exists that attenuated living tubercle bacilli introduced into the human organism can under certain conditions reacquire their earlier virulence. For this reason he believes it hazardous to use attenuated living material; and therefore prefers Schroeder's vaccine, which consists of killed tubercle bacilli which have, however, not lost their immunizing properties. The preparation was used every three weeks and in doses of 0.0025 to 0.002 mgm. bovine tubercle bacilli: 130 cases were treated with favorable results. Clinically and roentgenologically there was a definite retrogression of the tuberculous process, with disappearance of clinical symptoms and improvement decidedly greater than in the nontreated cases. In a majority of the treated cases tubercle bacilli disappeared from the sputum and the blood-picture improved.—*Die Behandlung der Lungentuberkulose mit der Schröderschen Vaccine, R. Brinkmann, Ztschr. f. Tuberk., 1930, lviii, 145.*—(*H. J. C.*)

Treatment of Tuberculosis with Living Tubercle Bacilli.—It is well established by animal experiments that living tubercle bacilli produce a better immunity than dead ones, and that the degree of immunity is roughly parallel with the degree of virulence of the bacilli. In previously infected animals the danger of reinfection depends on the dosage of reinfection and on the mode of administration. Cutaneous or intracutaneous reinfections are least dangerous. It is a common clinical experience that visceral tuberculosis is favorably influenced by the occurrence of a cutaneous tuberculosis. All these facts together form the rationale of the treatment of tuberculous patients with cutaneous or intracutaneous injections of virulent living tubercle bacilli. About 70 patients have been treated with a total of some 500 injections. No ill effects have been observed beyond the formation of local reactions and occasional small cold abscesses. Focal reactions are less likely to occur than with tuberculin treatment. The indications and contraindications are similar to those for tuberculin treatment. But this treatment has a somewhat wider field of application than tuberculin treatment because of the lesser danger of focal reactions. The therapeutic results are claimed to be satisfactory but are not related in detail.—*Tuberkulosebehandlung mit lebenden Tuberkelbazillen, H. Kutschera-Aichbergen, Wien. klin. Wchnschr., December 11, 1931, xliv, 1556.*—(*M. P.*)

Treatment of Pulmonary Tuberculosis with Convalescent Blood.—Treatment with convalescent blood obtained from cases that had recovered from acute tuberculosis is thought to transfer immunizing properties. In addition, the nonspecific therapeutic action of the blood as a stimulating and hormone effect is given consideration. Sixty-three cases were treated, and, of these, 56 were predominantly productive and 7

predominantly exudative; 36 were open cases of tuberculosis and 27 closed; and 15 cases were definitely progressive and 48 of stationary character. Treatment extended over a period of 1 to 2 months up to one-half year or more. Blood was given every 5 to 8 days in from 1- to 10-cc. amounts intramuscularly. In most cases defibrinated blood was used. Of the 63 cases, 47 showed decided improvement, 18 slight improvement, 2 remained unchanged and 2 progressed: 14 became free from bacilli. Eleven cases were treated with the Uhlenhuth tuberculosis cow-serum without showing any significant results.—*Über die Rekonvaleszentenblutbehandlung bei Lungentuberkulose, J. Leitner, Beitr. z. Klin. d. Tuberk., 1930, lxxvi, 174.*—(*H. J. C.*)

Treatment of Tuberculosis with Thanatophthisin.—*Thanatophthisin* is a serum obtained from horses treated with extracts of tuberculous lesions from spontaneously infected cattle. For the purpose of immunization the horses are given gradually increasing doses of the material subcutaneously. The preparation has been used on 93 sanatorium cases and 10 others since 1926. Most of the cases were advanced. In a number of the cases pneumothorax and phrenic exairesis were employed in addition to the serum. The preparation is harmless and acts as an irritant and stimulation therapy. Results were favorable.—*Klinische Beobachtungen und Erfahrungen bei der Behandlung von Tuberkulosekranken mit "Thanatophthisin," E. Hager and W. Behrendt, Ztschr. f. Tuberk., 1930, lvi, 97.*—(*H. J. C.*)

Drug Treatment of Haemoptysis.—Pulmonary haemorrhage is treated at the beginning by giving a teaspoonful of equal parts of fluid extract of *hamamelis virg.* and *calcium bromati* in milk or water. Eventually there is added *oleum menthae, anaesthesin,* and *solution suprarenin* 0.1 per cent. In the interval the following concoction is used: *decoct. gelatin album 150, Adalin 3, calcii bromat. (or lactate 10) 10, atropin sulphate 0.0075, codein phosphate 0.3 (or Dicodid or Eucodal), papaverin chloride 0.5–0.8, Tc. digital. titr. 6 to 8.* This is given three times a day in teaspoonful amounts.—*Beitrag zur Therapie von Lungenblutungen bei Tuberkulose, H. Erdmann, Ztschr. f. Tuberk., 1930, lvi, 449.*—(*H. J. C.*)

Medicinal Treatment of Fever in Chronic Tuberculosis.—Subacute and chronic cases of pulmonary tuberculosis in which collapse therapy is contraindicated or impossible often run a persistent febrile course. Artificial reduction of fever with the ordinary antipyretic drugs is not always easily accomplished, and often provokes profuse sweats which are noxious to the patient and damaging to the circulation. Quinine hydrochloride (0.1 gm.) three or four times daily is serviceable in such cases and leads to a decrease of temperature, without sweating. It has also a stimulating effect. It proves helpful when combined with cardiazol in 0.05 gm. doses.—*Zur Behandlung fieberhafter Lungentuberkulosen mit Cardiazol-Chinin (Knoll), Grünewald, Deutsche med. Wchnschr., February 27, 1931, lvii, 364.*—(*H. S. W.*)

Lipoid Therapy in Tuberculosis.—Gamelan, a preparation containing various lipoids in suspension, was used subcutaneously in the treatment of tuberculosis. Good results are reported in all of the 12 cases studied. Its action is to stimulate body-defense mechanisms against the tubercle bacilli.—*Contributo alla terapia lipoidica della tubercolosi, A. Schweitzer, Tubercolosi, July, 1930, no. 7, 292.*—(*U. E. Z.*)

Treatment of Tuberculosis with Santuben.—The author reports on the use of *Santuben* on 15 patients, the treatment being carried over from 3 to 7 months, in which he found a favorable influence on the general condition, the temperature and the amount of sputum, as well as a tendency toward resorption of serous effusions in specific peritonitis and exudative pleuritis. In no case was a focal or general reaction observed. Santuben is a guaicacolsilicum-combination tuberculin ointment suggested by Wolff-Eisner, which is supposed to stimulate the formation of antibodies in the skin. The author concludes that Santuben is a practical and harmless form of tuberculin of value in treatment.—*Die Behandlung der Tuberkulose mit Santuben nach Prof. Dr. A. Wolff-Eisner, A. Eckstein, Ztschr. f. Tuberk., 1931, lix, 350.*—(*H. J. C.*)

Spasmodic Asthma.—Although spasmodic asthma may have numerous excitants such as psychical stimuli, foreign proteins and peripheral stimuli, the latter including nasal irritation, eyestrain, hay fever, sinus infections, gastric and intestinal disturbances, the author attributes a very large share of the production of asthma to the peripheral stimulation exercised by nasal abnormalities. The disappearance or mitigation of the asthmatic paroxysm after the removal of nasal disturbances is now a frequent observation. Usually there is a recrudescence of the asthma after the recurrence of the nasal disease. The disappear-

ance of sensitiveness to special irritants after operation is also noticed. It is obvious that asthma depends on many diseased conditions and is a symptom rather than a disease. The practitioner must analyze it and attack it from various angles, but any nasal condition should be thoroughly investigated and judiciously dealt with.—*The Nasal Element in Spasmodic Asthma, J. Dundas-Grant, Brit. M. J., December 19, 1931, no. 3702, 1127.*—(*M. E. P.*)

Atropine Test in Asthma.—Since atropine prevents nervous stimuli from reaching the bronchial muscle, it has been thought that it might be used to differentiate between bronchoconstriction due to reflex stimulation of the vagus and that due to direct stimulation of the bronchial muscle; that is, between nervous and chemical stimulation. As a method of estimating the part played by nervous stimuli in inaugurating bronchospasm, a full dose of atropine might be injected subcutaneously at the onset of an attack. In those cases in which contraction of the bronchial muscle is due to direct chemical stimulation of the muscle, atropine would be expected to have no effect, whereas in the truly nervous type the attack would be aborted. In this way the patients whose attacks are due to psychical stimuli alone could be differentiated from those who owe their symptoms to a fundamental chemical alteration of the blood.—*An Atropine Test in Asthma, M. Gillespie, Brit. M. J., August 29, 1931, no. 3686, 384.*—(*M. E. P.*)

Infectious Asthma and Sinusitis.—In the study of the asthma problem in adults, the allergic enthusiast may be confronted with an impasse in at least 47 per cent of cases if he attributes the attacks essentially to the phenomenon of protein hypersensitiveness. Increasing clinical experience in the study of the adult asthmatic patient leads to the conclusion that investigation for atopy is only one means of differentiating that from other commonly encountered forms. A broader view lies in the conception that there exists in asthmatic patients a constitutional imbalance of the vegetative nervous system, referable especially to the respiratory tract, in which an allergic factor may coexist and be one of the many exciting agents of the paroxysm. When the allergic excitant is absent other stimuli, particularly infections of the respiratory tract as held by older clinicians, may be responsible and play the dominant rôle in initiating the attacks. Similar infections in normal individuals do not evoke asthmatic paroxysms, just as atopens in atopic individuals, whose autonomic imbalance is not referable to the respiratory tract, may elicit skin or gastrointestinal symptoms rather than asthma. The extent of damage to the tissues of the nose, sinuses, bronchi and alveolar passages following infection have been repeatedly demonstrated by histological studies of autopsy material in asthmatic patients. A comparative study of pathological changes in fatal bronchial asthma in nonsensitive patients showed that there were striking similarities in these cases to the pathological changes presented in fatal allergic cases. In summarizing these observations one is forced to ask whether infection in the sinuses or lungs in an individual with a constitutionally imbalanced vegetative nervous system does not evoke the same mechanism as in true allergy. Out of a group of 409 cases of bronchial asthma in adults, 200 were found to be nonsensitive. Of this number, 132 were completely investigated from the point of view of the presence of allergy, as determined by skin tests, and infection as established by clinical and roentgen studies of the nose, sinuses and chest. The underlying causes revealed by these investigations pointed to the fact that the asthma in eighty-seven was primarily due to sinus disease, the ethmoids and the antrums being chiefly implicated, while clinically the lungs showed secondary bronchitis with no distinctive roentgen finding. Twenty-two patients had unresolved pneumonia, 4 cases were complicated by bronchiectasis, and 21 patients had sinus disease associated with unresolved pneumonia. In the group that comprised this report, nineteen were under observation for from three to four years. Of these, the youngest was 17 years of age and the oldest 60. The duration of the disease was from one week to twenty years. Many had no knowledge of the fact that they had pulmonary infections and attributed the onset of their attacks to an ordinary cold or grip. None of the patients had radical surgical intervention. The treatment was largely that of antral washings and topical applications to the ethmoids. Compared with incomplete and questionable nasal surgery, these patients were able to do as well if not better. —*Infectious Asthma and its Relationship to Chronic Sinus and Pulmonary Disease, J. Harkavy and F. Maisel, J. Am. M. Ass., May 30, 1931, xcvi, 1843.*—(*G. L. L.*)

Sinus Chest Infections.—The clinical symptoms of sinus chest infections may closely simulate active pulmonary tuberculosis. Râles and elevation of temperature are present. A careful history should prompt a roentgen examination of the sinuses in at least 9 per cent of chest cases examined.

Fluoroscopic examination of the chest has very limited differential diagnostic value in determining early tuberculosis and no value in distinguishing early tuberculosis from chronic infection. When a known sinusitis exists, a stereoscopic chest study is indicated, to determine the degree of pulmonary change due to chronic infection. These roentgenograms have immense comparative value for later examinations.—*Accessory Sinus Infection in Suspected Pulmonary Tuberculosis, J. D. Osmond, J. Am. M. Ass., December 12, 1931, xcvii, 1778.*—(*G. L. L.*)

Origin of Aseptic and Atraumatic Pleurisy.—So called "military pleuritis," next to beri beri, is the most disabling affliction of the Japanese soldiers. It is more frequent in Japan than in all other countries, while tuberculosis has about the same incidence as in other countries. This in itself bespeaks the nontuberculous nature of this pleurisy. The great frequency of the condition among recruits indicates that it is due to changes in living conditions. Initially it has no inflammatory character, but this occurs at a later date. To explain the condition the authors attempted to produce a primary or idiopathic (aseptic atraumatic) pleurisy by the intravenous injection of sympathicotonic-acting nerve poisons and coincident bodily exercise. Adrenalin, calcium chloride and glucose solution under these conditions appeared to cause pleural exudates, with death of the animal on the 4th or 5th day. Parasympathicotonic poisons, like acetylcholin and potassium chloride, caused no pleural changes. Preliminary treatment of the latter agents prevented the development of exudates by the foregoing first-cited reagent. With bilateral sympathectomy pleurisy did not occur following the use of the sympathicotonic agents.—*Experimentelle Versuche über die Entstehung der aseptischen und atraumatischen Pleuritis. Eine Beweisführung für das Wesen der sogenannten Militärpleuritis und der idiopathischen Pleuritis in Japan, T. Kanai and K. Minami, Ztschr. f. Tuberk., 1930, lvi, 434.*—(*H. J. C.*)

Pneumothorax by Aspiration.—A frequent and disagreeable complication of artificial pneumothorax therapy is undue shifting of the mediastinum. It is usually recognized by the observation that the intrapleural pressure does not increase while air is being introduced. Mediastinal shifting may occur with negative intrapleural pressures. In order to avoid this complication, filtered air was introduced into the pleural cavity by simple aspiration instead of using the usual pneumothorax apparatus. As long as the amount of air introduced is so controlled entirely by the patient's respiration mediastinal shifting does not occur. When this method is used the patient may not lie on the contralateral side because this in itself tends to produce mediastinal shifting; the insufflation must be done with the patient lying on his back. This method is particularly advisable in patients with movable mediastinum and with a sensitive circulatory system. In animal experiments it was found that irritating material introduced into a pneumothorax cavity produces considerably more inflammatory, and eventually fibrotic changes in the parietal pleura than in the visceral pleura. This is explained by the fact that an increased intrapleural pressure decreases the lymph stream in the parietal structures and tends to increase the lymph stream in the lung. It seems therefore justifiable to produce inflammatory reactions in a pneumothorax cavity with the intention of fibrosing the parietal pleura. A parietal pleura altered in such a way resorbs air much less readily than a normal one, and therefore the intervals between insufflations can be prolonged and the intrapleural pressure is less subject to marked changes.—*Ueber Aspirationspneumothorax, J. Sorgo, Wien. klin. Wchnschr., October 30, 1931, xliv, 1369.*—(*M. P.*)

Spontaneous Haemopneumothorax.—A case is reported of a man, 44 years of age, who, after a violent attack of coughing, complained of severe pain in the left chest. During the ensuing two days he showed increasing signs of an internal haemorrhage and pneumothorax and died on the third day. The postmortem examination revealed a completely collapsed left lung, with displacement of the heart to the right, and the left pleural sac filled with blood. At the apex of the lung were two firm, cord-like adhesions, which appeared to have been torn away from the chest-wall before death. Careful search failed to reveal in the lung the site of its rupture or of the haemorrhage, but it seemed quite possible that it was from the torn adhesion that bleeding took place.—*Spontaneous Haemopneumothorax with Unusual Post-Mortem Findings, E. G. Housden and A. Piggot, Brit. M. J., November 21, 1931, no. 3698, 941.*—(*M. E. P.*)

Origin of Fibrin Bodies in Pneumothorax.—Rare, free, fibrin bodies in pneumothorax are considered by many as due to haemorrhage. In two of the author's cases it was shown that the presence of an exudate is necessary for the formation of fibrin balls, and that these fibrin bodies can only occur with small exudates, when a pneumothorax

lung dips into and slowly grows away from the fluid. Thus a fibrin string, hanging from the lung, forms, and this later tears off and takes on a ball shape with later organization. Serial examinations revealed this mode of development and haemorrhage did not come into consideration.—*Zur Entstehung der Fibrinkörper im Pneumothoraxraume, S. Pollak, Beitr. z. Klin. d. Tuberk., 1930, lxxiv, 494.*—(*H. J. C.*)

Mediastinal Displacement in Pneumothorax.—Displacement of the entire mediastinum or of its superior-anterior or inferior portions may occur in spontaneous or therapeutic pneumothorax and is due to a disturbance of the normal intrathoracic equilibrium. Bulging of the superior-anterior mediastinum is observed in therapeutic pneumothorax and occurs only during expiration. It occurs usually in pneumothorax of the right side, the bulging invading the left hemithorax. Displacement from left to right in pneumothorax of the left side is rare, because of the position of the oesophagus. Fixation of the mediastinum through changes in the mediastinal pleura occurs in older adults and in long-standing cases of pulmonary tuberculosis; consequently displacements are most frequently observed in young adults. The condition results in slight anteroposterior, but no lateral compression of the contralateral lung, and occurs in the presence of negative intrapleural pressure. Infrequently, in pneumothorax of the right side, the right upper lobe bulges into the left chest through the superior anterior mediastinum. It carries with it a hernial sac consisting of mediastinal pleura, becomes fixed, and is uninfluenced by respiration. This type usually is associated with a relatively nonelastic mediastinal pleura and with positive pressure in the pneumothorax, and may cause respiratory embarrassment, thus differing from the mobile type of displacement. The degree of displacement depends upon the mobility of the mediastinum and its contents, and upon the degree of pressure in the pneumothorax. A mobile displacement aids, and a fixed displacement hinders oxygenation of the contralateral lung. In the absence of pleuropericardial adhesions, the heart and the entire mediastinum may be displaced into the contralateral hemithorax. This condition is observed in spontaneous pneumothorax with positive pressure, usually associated with pleural exudate. The rapidity of displacement determines the acuteness of symptoms. This type of displacement may occur to the right as well as to the left, though the left to right displacement requires greater pressure and the degree of displacement is less. The mediastinum tends to become fixed in the displaced position in these cases, and this is accompanied by respiratory embarrassment. Expiratory encroachment of the entire mediastinum upon the contralateral lung without respiratory embarrassment is noticed in young adults. In these cases also the mediastinum tends to become fixed. Mediastinal displacement may allow the introduction of considerable quantities of air without change in manometric readings. Functional symptoms are more apparent when pressure is applied to a relatively fixed mediastinum than when there is considerable deviation of a flexible mediastinum under negative pressure. The author was unable to demonstrate paradoxical diaphragmatic breathing in artificial pneumothorax. Immobilization of the chest-wall of a hemithorax of a normal person caused no movement of the mediastinum during respiration. Movement of the mediastinum was noticed during forced respiration in a person with a paralyzed diaphragm. The hemithorax on the side of the paralyzed diaphragm was immobilized and then the mediastinum remained stationary during forced respiration. —*Mediastinal Displacement in Pneumothorax, W. C. Pollock, Ann. Int. Med., October, 1931, v, 486.*—(*R. M. F.*)

Mechanism of Tension Pneumothorax.—The author reports two instances in which a tension pneumothorax and subcutaneous emphysema followed a thoracocentesis. In order to infiltrate the subcutaneous tissue with air, a pressure greater than atmospheric is required, for the skin is at all times subject to the latter pressure. Forced expiration, especially with momentary closure of the glottis as in coughing, grunting, talking and straining, produces a marked increase of the pressure in the pneumothorax. For the development of a tension pneumothorax, a valvular perforation of the lung is required, through which air can pass only from the lung into the pleural cavity, any retrograde current immediately collapsing the opening in the soft pulmonary tissue. If the perforation were to remain patent to air currents in either direction, an average pressure above atmospheric pressure could not be obtained. By means of a valvular perforation, air is aspirated into the pleural cavity with each expansion of the chest in inspiration and remains trapped during expiration. The result is a gradual collapse of the elastic lung. Since the alveolar air is in free communication with the external air through the respiratory tract, its pressure in inspiration is one atmosphere or less, depending on the vigor of the inhala-

tion. Therefore, air can escape into the pleural cavity during inspiration only until the inspiratory pressure in that chamber becomes atmospheric. This mechanism does not account for the production of a tension pneumothorax in which a positive intrapleural pressure is obtained during inspiration as well as expiration. Since the bronchial passages communicate with each other through the trachea, an analysis of the part played by the intact lung may provide an adequate explanation. The pneumothorax side of the chest becomes relatively splinted in comparison with the opposite side, which now has, in compensation, exaggerated respiratory movements. If the larynx is momentarily closed during expiration, as in coughing or grunting, air can be forced out of the intact lung into the splinted pleural cavity through the intervening bronchi and valvular perforation. In order for the intact lung to function in pumping air into the pneumothorax, the expiratory muscles on that side must exert more force than those on the side of the pneumothorax. That the latter force is less may be inferred from the splinting, which is readily observed clinically. Multiple small skin incisions are generally recommended for the relief of the subcutaneous emphysema. Unless the pneumothorax causes definite embarrassment, it is advisable not to aspirate, since the increasing pressure in this chamber tends to prevent the further admission of air and thereby facilitates the healing of the perforation. To alleviate coughing and grunting, the liberal administration of morphine is advised. Aspiration is indicated when the patient becomes embarrassed.—*Tension Pneumothorax with Subcutaneous Emphysema: the Mechanism of Tension Pneumothorax, N. M. Fenichel, J. Am. M. Ass., July 4, 1931, xcvii, 20.*—(*G. L. L.*)

Kienboeck's Phenomenon in Pneumothorax.—In 1898 Kienboeck noticed radioscopically in a patient suffering from pyopneumothorax that the level of the fluid became higher during inspiration and lower during expiration. At first he attributed the elevation of the fluid level to an inspiratory contraction of the diaphragm. Later he admitted a paralysis of the hemidiaphragm on the affected side, and advanced his theory that the paradoxical movements were due to the increased intraäbdominal pressure incident to the inspiratory descent of the hemidiaphragm on the unaffected side. Various other authors have studied the same problem and have attributed the phenomenon to a hypotonicity of the diaphragmatic muscle on the diseased side. Following a series of carefully controlled studies, the author found that in cases of hydropneumothorax Kienboeck's phenomenon may be due to rythmic pendulum-like movements of the mediastinum. The mediastinum moves toward the pneumothorax during inspiration and recedes during expiration. In these cases the diaphragm played no part, and changes in the endopleural pressure caused no variations in the extent of the movements. The paralyzed diaphragm may play an important rôle in its production, but it is always secondary to a pulling force exerted by the lung itself. Costodiaphragmatic adhesions also give rise to similar changes in the fluid level without mediastinal fluctuations. Elevated positive endopleural pressure plays a secondary rôle. Any of the above-mentioned factors may act concomitantly. The retracting force exerted upon the intrathoracic structures by the lung itself is the principal cause.—*Contributi di meccanica respiratoria in pneumotorace. Sulla patogenesi del fenomeno di Kienboeck, D. Maestrini, Tubercolosi, December, 1929, xxi, 507.*—(*U. E. Z.*)

Roentgenology of Lobar Pneumonia.—The roentgenological findings in 120 cases of lobar pneumonia, 50 of which were seen in the early stages of the disease, are described. In all of the early cases cone-shaped consolidations, with the base of the cone toward the periphery, were demonstrable. The triangular shadows whose bases were directed laterally were best shown in the anteroposterior view, and appeared as rounded densities in the lateral view. Thirteen cases were studied in the anteroposterior view and revealed central-type shadows. In all of these cases the shadow was due to a posterior triangle, with the consolidation limited to the lower lobe. In 5 cases the process later showed extension into the lateral periphery of the involved lobe, while in the other 8 the consolidation remained limited to the medial portion of the lung. The earliest roentgenological findings were not those of a homogeneous triangle, but of a group of soft linear shadows arranged in a triangular manner separated from each other by the air-containing bronchi.—*Roentgenologic Studies in Early Lobar Pneumonia, W. H. Ude, Am. J. Roentgenol. & Rad. Ther., November, 1931, xxvi, 691.*—(*W. I. W.*)

Metabolism in Pneumonia.—The literature on the excretion of chlorides in pneumonia is briefly reviewed. This shows that its diminished excretion in the urine is not due to loss in the perspiration or in the faeces, and that the retention cannot be accounted for by a simultaneous retention

of water, by deposition in the lungs, or by an increased concentration in other tissues. The symptoms following the intratracheal or intrapulmonic injection of pneumococcus cultures in dogs are described. Shortly after such injection, the amount of chloride in the urine is greatly increased. This is followed by a subnormal excretion. From a review of the literature it is shown that the same sequence is observed in malaria in man. It is suggested that the low concentration of chloride in the plasma in pneumonia is due to a rapid excretion of chloride in the early stages of the disease. Data from the literature are shown to support this view, while none could be found to oppose it. Calculation of the changes observed in the patients showing the greatest recorded retentions of chloride in pneumonia show that the retention can be satisfactorily accounted for in terms of the hypothesis here presented. The clinical significance of the results is briefly discussed.—*Metabolism in Pneumonia, I. Greenwold, Arch. Int. Med., September, 1931, xlviii, 418.*—(*W. I. W.*)

Treatment of Lobar Pneumonia with Immune Blood.—Passive immunity was produced in normal men by intramuscular injections of pneumococcus vaccine, and their blood used in the treatment of lobar pneumonia. The object was to determine what effect the combination of type-specific pneumococcus antibodies plus uninjured complement present in normal human blood might have in arresting severe pneumonic infection. Of 8 cases treated by immune transfusion, 6 were due to pneumococcus type III and 2 to pneumococcus type II. Four of the 6 type-III cases had a bacteraemia, and all died. Two of the type-III cases with sterile blood-cultures survived. Of the 2 cases of type-II pneumonia with positive blood-culture 1 died and 1 survived. No favorable effect was observed on the clinical condition of the patients as a result of any of the transfusions. The introduction of protective substances present in the donor's blood could not be demonstrated in the patient's blood in the 5 fatal cases, and appeared to have no influence on the course of the disease in the 3 recovered patients.—*Immune Transfusion in Lobar Pneumonia, A. L. Barach and M. Soraka, Am. J. Med. Sc., December, 1931, clxxxii, 811.*—(*W. I. W.*)

Postpneumonic Lung.—Chronic lung changes following pneumonia depend on the degree to which the various tissues of the lung are involved by the acute process and the severity of the injury which they sustain. Those pneumonias in which the connective-tissue framework of the lung, the walls of the bronchial tree, and the vascular and lymphatic channels are seriously damaged or destroyed, exhibit the largest number of chronic pulmonary sequelae. Among the most important of the pneumonias likely to result in chronic changes are influenzal pneumonia, influenzal pneumonia invaded by streptococci, the bronchopneumonias accompanying measles and pertussis, and Friedländer's pneumonia. The pneumonic process may take any of three possible courses: (1) the exudate may be absorbed and recovery ensue, (2) the exudate may be organized, and fibrotic changes and chronic nontuberculous infections develop, or (3) suppuration may occur. If either of the two latter possibilities eventuate, chronic terminal conditions may be expected. These may take the form of bronchial dilatation and bronchiectasis, chronic nontuberculous infections, abscess-formation, or fibrosis, massive or diffuse in type. In the light of the modern conception of the muscular and elastic tissue of the bronchi and lungs, it appears that diffuse fibrosis following pneumonia may cause very considerable impairment of respiratory function and physical fitness. Roentgenography offers an available and accurate method of following pneumonia cases through convalescence and determining what percentage of these patients develop chronic pulmonary changes. The roentgenologist has the power to occupy the same position in the study of the postpneumonic lung in the living patient that the pathologist does in the investigation of pulmonary change in the cadaver. Systematic follow-up and periodical roentgen examinations of patients in whom resolution does not take place promptly would throw much light upon the incidence of chronic postpneumonic lesions.—*The Postpneumonic Lung, A. L. Hart, Am. J. Roentgenol. & Rad. Ther., September, 1931, xxiv, 371.*—(*W. I. W.*)

Phrenicectomy in Bronchiectasis.—In 17 cases of bronchiectasis,phrenic avulsion gave the following results: complete relief of symptoms in 4 cases; lasting improvement in 7 cases; and temporary improvement, with relapse, in 6 cases. Only in strictly basal and unilateral cases of bronchiectasis can phrenic avulsion be reasonably expected to relieve the symptoms completely. Temporary improvement is the rule if the lesions extend into the upper or midzone of the lung. Little benefit is to be hoped for in bilateral basal bronchiectasis with lesions in the upper or midzones.—*Indications for Phrenic Avulsion in Bronchiectasis, W. G. Oakley, Brit. M. J., August 29, 1931, no. 3686, 378.*—(*M. E. P.*)

Giant Growth of Pulmonary Artery with Congenital Bronchiectasis.—The postmortem examination of the body of a man, 69 years of age, who died following an operation for squamous-cell carcinoma of the penis, revealed an overgrowth of a branch of the left pulmonary artery. This was found in the lower lobe, and measured 6 cm. in length, with its diameter tapering from 8 to 3 mm. The wall was 1.5 mm. thick and was studded with atherosclerotic plaques. The artery was associated with a congenital bronchiectatic cavity, which cranially lay ventral and lateral to the artery, shifting its position caudally to lie anterior and medial to it. The condition represented by the artery was due to a congenital anomaly. It probably originated in a persistent branch of the postbronchial arterial plexus. The accompanying bronchiectasis resulted from the pressure exerted by the artery on the surrounding alveoli, which compressed them and caused ectasia of the bronchus due to lack of opposition.—*Isolated Giant Growth of a Branch of the Pulmonary Artery Associated with Congenital Bronchiectasis, S. R. Rosenthal, Arch. Path., September, 1931, xii, 387.*—(*R. D.*)

Treatment of Empyema.—Whatever the method of treatment of pleural suppuration employed, no cure is possible until the source of infection of the cavity ceases to act as such. If open operation is done in such a case, prolonged drainage and thick-walled sinus-formation results, requiring later extensive surgery. During the World War it was found that the average mortality rate was 30 per cent, and in some hospitals as high as 70 per cent. The rate was higher when operation immediately followed diagnosis. Of recent years many methods of closed drainage have superseded the incision and drainage method. The author presents the results of his method of treatment, aspirating through a large needle and replacing an equal amount of air, in a series of 35 cases, only 2 of which died. The aetiology was lobar pneumonia in 20 cases, influenza 5, gunshot wound 1, stab wound 2, unclassified pulmonary disease 4, pelvic infection 1, perinephric abscess 1, uncertain 1. There was not a serious infection of the chest-wall resulting from the many needle punctures. A point in the intercostal space corresponding to the lowest point of the empyema cavity is anaesthetized with 0.5 per cent solution of procaine hydrochloride. A large needle is now connected to a 50- or 100-cc. Luer syringe and inserted. A syringeful of pus is aspirated, the tube is clamped with haemostatic forceps, the syringe is disconnected, emptied, filled with an equal quantity of air, and injected. The amount of pus removed has varied from a few cubic centimetres to as much as 3,000 cc. at one time. The procedure is repeated as often as the fluid accumulates, on an average about every six days. If the fluid is gradually replaced with air as it is removed, the cavity maintains its original size and shape, and every drop of the fluid can be removed with practically no discomfort to the patient. Complete emptying of the cavity is essential to bringing about a cure. One patient was cured with one aspiration. The average has been about four. In the average case of this series only from 100 to 300 cc. of pus accumulated in an average period of six to eight days. This method has several advantages over the usual other means: there being practically no trauma to lung or pleura, the pleural surfaces may finally be free again and not adherent, as must be the case when open thoracotomy and drainage have been done. There being no drain, there is no secondary infection introduced from the outside. The patient is spared an operation, which itself has a certain percentage of mortality.—*The Treatment of Empyema by Aspiration and Air Replacement without Drainage: Review of Thirty-Five Cases, J. A. Danna, J. Am. M. Ass., May 2, 1931, xcvi, 1453.*—(*G. L. L.*)

Mortality in Acute Empyema.—This study opens with a historical survey of the treatment and mortality of empyema from Hippocrates to the present day. Whether the opened or the closed method for evacuation of pus is the safer is carefully discussed. The conclusion is reached that one or both methods may be used according to the clinical severity and to the type of infecting organism. The closed is the better method in the larger percentage of cases. Ninety-nine cases are reported with a total mortality of 6.1 per cent. Infants show a higher mortality rate than older children and young adults, but after fifty years of age the death-rate rises as high as 50 per cent.—*The Mortality of Operations for Acute Empyema, G. P. Muller, J. Thorac. Surg., October, 1931, i, 15.*—(*J. G. K.*)

Mediastinal Emphysema.—This condition occurs more frequently than the available literature would lead one to believe. The symptoms are constant and if kept in mind may lead to early recognition with a possibility of saving life. Possible causes include injuries and diseases of the lung, operations on the lung, perforations of the chest-wall, artificial pneumothorax, perforating wounds of the larynx, trachea and bronchi, wounds of the neck and diaphragm,

rupture of the oesophagus, and rupture of the stomach. The air travels along definite fascial planes and produces increased mediastinal pressure. The outstanding effect is a compression of the large veins entering both sides of the heart, the outward signs being due to this. The characteristic symptoms and signs include extreme cyanosis and dyspnoea, rapid pulse, congestion of the superficial veins of the neck, low blood-pressure, substernal discomfort and pain, and subcutaneous emphysema. There may be tympany over the sternum, heart-sounds may be distant, or emphysematous clicks synchronous with the heart-beat may be heard. The X-ray may show increased vascular markings in the lungs, or dark longitudinal streaks in the mediastinal shadow. Treatment must be aimed at (1) controlling the source of the air by closure of the rent in the lung or the bronchus, and (2) giving an outlet to air already accumulated by relieving a pneumothorax by insertion of a needle attached to tubing with a water seal, or relieving subcutaneous emphysema by incision of the skin. Incising the skin about the neck with relief of emphysema has been followed by recovery in several instances.—*Mediastinal Emphysema, P. M. Jessup, Arch. Surg., November, 1931, xxiii, 760.*—(*R. D.*)

Causes of Massive Collapse.—Neither the aetiology nor mechanism of massive collapse has yet been explained satisfactorily. Recent theories consider that posture may be a causative factor or that a combination of factors, such as the inhibition of the cough reflex, with impairment of respiratory function and a resulting accumulation of secretions, and blocking of the bronchi lead to atelectasis. Usually the condition develops during the first few days following operation. The patient complains of sharp pain in the chest, and cough. The temperature is elevated and respirations are increased and embarrassed. The condition may simulate pneumonia closely and may be diagnosed as such. Roentgenological examination will show dense pneumonic consolidation, elevation of the diaphragm, and displacement of the heart and trachea toward the involved side. The best treatment is to require the patient to cough and to have him lie on his side with the involved lung uppermost. If postural treatment does not reinflate the lung, bronchoscopy is indicated. Collapse of the lung due to tumor of the bronchus can be differentiated from idiopathic massive collapse only by bronchoscopic examination. A case of primary carcinoma of the bronchus, studied during several attacks of collapse, was observed to collapse after each reinflation. Spontaneous pneumothorax may be caused by rupture of emphysematous blebs on the surface of the lung. Usually the patient recovers rapidly, with absorption of the air in a few days, but many years may elapse before recovery is complete.—*Conditions Which Result in Collapse of the Lung, R. P. Potter, Radiology, August, 1931, xvii, 271.*—(*T. O. R.*)

Experimental Atelectasis.—Van Allen and Adams have attributed pulmonary atelectasis to the association of bronchial obstruction with labored respiration. The requirement of the latter factor is directly contrary to the observation of others in this field. Moreover, the statement was based on the use of an artificial obstruction in the bronchi in experiments running 24 hours or less and on the observation that obstruction of an air-passage to part of a lung caused no atelectasis. In the experiments here recorded dogs were used. Stenosis of the bronchus was produced by cauterization with silver nitrate. In the first group of animals 50 per cent solution of silver nitrate was applied for 10 seconds to the origin of the bronchus of the right middle lobe. The animals were killed in 1 to 6 weeks. If complete stenosis was present 100 per cent atelectasis resulted, but if an opening of only 1 mm. remained there was no atelectasis and the lobe appeared normal grossly and microscopically. In the second group a similar solution was applied within the bronchus, causing stenosis of only one of two main divisions. No atelectasis resulted, although the experiments ran 2 to 8 weeks. In 6 animals the cauterizing agent was applied to the bronchus of the accessory lobe. Respiration afterward was essentially normal in character. On autopsy the bronchus was stenosed and 100 per cent atelectasis was present. Careful anatomical studies have indicated that there are no communications between the air-passages of the lobules within one lobe of the lung. In view of these experiments it appears probable that there are communications between the alveoli of the many lobules of a lobe of the lung. *Conclusions: 1:* Complete obstruction of the air-passages of an entire pulmonary lobe was essential to the production of massive atelectasis. *2:* Complete obstruction of the air-passages of part of a lobe was not accompanied by atelectasis of the obstructed part. *3:* No particular form of respiration was essential to the production of massive collapse. (The rate of production may be increased by a valvular obstruction and a straining type of respiration.) *4:* Communications appear to

be present between the alveoli of adjacent lobules of a pulmonary lobe in dogs. *5:* Pulmonary suppuration was not associated with obstructive pulmonary atelectasis produced experimentally in dogs.—*Obstructive Pulmonary Atelectasis, W. E. Adams and H. M. Livingstone, Arch. Surg., September, 1931, xxiii, 500.*—(*R. D.*)

Impression of Pulmonary Artery on Oesophagus.—The impressions of the aorta, the pulmonary artery, and of the left auricle upon the barium-filled oesophagus can be visualized with the roentgen-ray. Visualization of the oesophagus is employed in the study of disease of the oesophagus itself as well as conditions which produce pressure upon it. The relation of the oesophagus to the aorta has been used for measuring the diameter of the aorta. The impression of the aorta upon the oesophagus is visible in the anterior views and in the first oblique position. The displacement of the oesophagus produced by the enlarged left auricle in mitral stenosis is almost a pathognomonic sign and is seen better in the first oblique position, as is the impression of the pulmonary artery on the oesophagus. It lies between the aortic impression above and the auricular impression below. It is often seen in some degree in healthy persons, but is modified or exaggerated by disease, such as emphysema and other chronic pulmonary affections, with enlargement of the pulmonary artery and its branches. The anatomy and relations of the oesophagus, heart and great vessels are reviewed. The so called pulmonary-artery impression is made by a combination of the left bronchus, and the right branch and main-stem of the pulmonary artery. In general the size of the pulmonary varies inversely with that of the aorta. In the interpretation of these oesophageal impressions it is well to remember that a favorite site of carcinoma of the oesophagus is the region of the left bronchus. Roentgenograms and diagrams are used to illustrate the points discussed.—*The Pulmonary Artery Impression on the Esophagus, J. Parkinson and D. Bedford, Lancet, August 15, 1931, ccxxi, 337.*—(*R. M. F.*)

Occlusion of Pulmonary Artery.—Only 5 cases of complete thrombotic occlusion of the pulmonary artery or one of the major branches occur in the literature. These cases are summarized. Thrombosis of the smaller arteries is fairly common. Brenner recently concluded, from 6 cases of thromboses of large branches, that the thrombosis usually occurs after severe congestive failure has set in. In the case here reported the clinical diagnosis was as follows: Arteriosclerotic and hypertensive heart disease with dilatation and marked hypertrophy of the heart, aortic valvular disease with regurgitation, congestive failure, chronic passive congestion, and terminal bronchopneumonia. At necropsy the most striking finding was a complete thrombotic occlusion of the right pulmonary artery. The thrombus showed no evidence of canalization. The right lung was smaller than the left, which was in a state of chronic passive congestion, but showed no evidence of atelectasis or necrosis. Compensatory circulation was provided by an hypertrophied bronchial artery and two other vessels of unusual size which anastomosed with it and apparently originated from the inferior thyroid arteries. In the right hilum was a mass of partly calcified and ossified fibrous tissue, which surrounded but did not obstruct the primary bronchial branches and the first branches of the pulmonary artery. The pulmonary arteries in corresponding peripheral portions of both lungs were occluded by fibrous plugs. The right lung contained less blood than normal, and the alveoli contained a small amount of mucus. There was no evidence of tuberculosis. The heart was greatly hypertrophied, and both ventricles were dilated. The aortic valve was bicuspid (evidently a congenital lesion), and the cusps were calcified, so as to produce both stenosis and insufficiency. Colloid goitres were found substernally and in the anterior-superior mediastinum, and there was a calculus in the right renal pelvis, but these conditions apparently had no bearing upon the lesion of the pulmonary artery. There was no evidence of syphilis. *Conclusions:* Total thrombotic occlusion of a main branch of the pulmonary artery may occur without damage to the lung, provided it develops slowly and allows collateral bronchial circulation to develop. Such a lesion is suggested by the presence of cyanosis and right-sided heart failure without other obvious cause.—*Total Occlusion of the Right Branch of the Pulmonary Artery by an Organized Thrombus, J. H. Means and T. B. Mallory, Ann. Int. Med., October, 1931, v, 417.*—(*R. M. F.*)

Passive Congestion of Lung in Coronary Sclerosis.—Three cases, two of which had a fatal termination, demonstrate an unusual type of passive congestion which always indicates the presence of severe coronary disease and left ventricular degeneration. Centrally there are heavy shadows to be seen in the film while peripherally and especially in the bases of the lungs the areas remain comparatively clear.—*Passive Congestion of the Lungs in the Presence of Coro-*

nary Sclerosis, J. F. Chapman, Radiology, August, 1931, xvii, 342.—(T. O. R.)

Diaphragmatic Hernia.—This condition is no longer a rarity. It is difficult to diagnose because of the complex symptomatology, often simulating other diseases of the thorax and the abdomen. The 45 cases here reported had previously been treated for cholecystitis (14 cases), gastric and duodenal ulcer (13 cases), intestinal obstruction (3 cases), and heart disease (4 cases). These herniae may be classified as congenital, acquired and traumatic; but for surgical usage the congenital type is unimportant as most infants with this deformity die soon after birth. The remaining cases may be classified as traumatic or nontraumatic. The most common location is at the oesophageal hiatus. Here a sac is generally present, and it rarely contains more than a part of the stomach, while in other locations there is usually no sac and the contents may consist of parts of stomach, small and large bowel, and even spleen and liver. The diagnosis is generally made or at least confirmed by roentgen-ray studies of the gastrointestinal tract. The treatment should always be surgical, unless radical operation is contraindicated by the general condition of the patient. A preliminary paralysis of the hemidiaphragm, by phrenicectomy, proved helpful in certain cases in reducing the hernia, and in closing the opening. In 4 patients only this procedure was done because of grave complications. All have shown slight to moderate clinical improvement. The abdominal, instead of the thoracic, approach seemed the better procedure in most cases, although in two of the series both methods were used. Thirty-eight patients were repaired surgically; five died within twelve days after operation; the rest are well, having been examined within the past six to twelve months.—*The Diagnosis and Treatment of Diaphragmatic Hernia, S. W. Harrington, J. Thorac. Surg., October, 1931, i, 24.—(J. G. K.)*

Pneumonoconiosis.—Pneumonoconiosis is a more or less necessary industrial risk, but its progress and serious aspects may be lessened by certain preventive measures in most instances. Silica in finely divided state is still generally conceded to be the harmful agent in most dusts which affect the lungs in any serious way. It is the active fibrosing agent in the condition which we recognize as essentially a pulmonary fibrosis, adherence to the general term of *pneumonoconiosis* still being advisable. Studies of different groups of workers exposed to various concentrations of dust revealed that 10 million particles, containing approximately 35 per cent of silica per cubic foot of air, could be tolerated without great injury to the lungs over a long period. Pneumatic tool-workers were exposed to from 55 to 64 million particles of dust per cubic foot, workers in cement plants to 26 million, hard-coal miners to 231.5 million and soft-coal cutters to 112.3 million. For comparison, municipal street dust in congested districts was found to contain 4.1 million, whereas in residential districts it contained 1.8 million particles. The pathological features are considered under the following headings: (1) the entrance of dust; (2) the "dust cell"; (3) entrance of the "dust cell" into the lymphatic system of the lungs as a carrier of silica or other particles; (4) influence of the deposited silica in the production of fibrous tissue; (5) the action of silica; (6) elimination of dust; (7) the predisposition of the fibrotic and silica-saturated lung to respiratory infections, especially tuberculosis. The roentgenological classification of pneumonoconiosis is described in three stages, designated numerically. The first stage has been characterized roentgenographically by a definite increase in the prominence and extent of the hilum shadows, an increased prominence and thickening of the trunk shadows and a greater prominence of the linear markings of the peripheral zone. Enlarged lymph nodes may be interpreted in the hilum region. The general appearances of this first stage are not characteristic of pneumonoconiosis alone, but may be simulated by many other conditions, such as, especially, passive congestion, acute or chronic respiratory infections, chronic bronchial catarrh and bronchiectasis. These first-stage appearances correspond exactly to the pathological changes of this period. The large numbers of dust cells that have reached the pulmonary lymph nodes institute more or less fibrosis and enlargement of these nodes; hence the dense and larger and more homogeneous hilum shadows. A lymphatic block in this direction is also instituted by the fibrosis in the lymph nodes. Lodgment of cells in the peribronchial and perivascular lymphatics causes, with the lymphatic block, the thickened trunks and prominent linear markings. The second stage has been characterized by the distinctive distribution of small rounded densities, varying in size from pinhead to pea, and distributed throughout both lungs. The nodules appear first on the right side, around the root of the lung, and usually become quite perceptible here before they are seen on the left side. The appearances of this stage are so characteristic that the condition

is not likely to be confused with any others except miliary tuberculosis, tuberculous bronchopneumonia, and certain mycotic infections. The fibrotic nodules have their incipiency in the lymphoid deposits. Some of these become larger and larger by aggregation of cells, their liberated dusts and subsequent fibrosis. They are the greatest primary centres of obstruction to lymphatic activity outside of the pulmonary lymph nodes. The third stage has been characterized by appearances due to a predominance of diffuse fibrosis which may present three fairly definite and distinct appearances, although all of them may be observed in the same case: *1:* The larger nodules of the second stage may coalesce into very much larger and irregular masses, or be found close together with more or less haze between them. *2:* A more or less diffuse fibrosis, somewhat similar in appearance to the fibrotic process representing the late result of an extensive bilateral chronic pulmonary tuberculosis: usually definite nodules are still present, although not always. *3:* The presence of massive fibrotic areas presenting the appearance of extensive pulmonary consolidations: in the late period of this stage, there is usually one such area on each side, rather symmetrically located subapically, but sometimes unilateral, or more extensive on one side than the other. The first two appearances result from an aggregation and coalescence of the smaller nodular fibrotic areas of the second stage or an advance of the interstitial fibrosis or both. The pathological identity of the areas of massive fibrosis is more or less problematical. Investigators of considerable experience, notably Mavrogardato and Watkins-Pitchford, believe they are due to a combination of silicosis and tuberculosis, although their origin from a coalescence of nodules is regarded as possible. These areas seldom caseate, but, when they do, bacilli are often found. Tuberculosis with silicosis is a progressive and serious disease, associated with larger nodules, asymmetrically distributed and composed of young connective-tissue elements, among which tubercle bacilli are found. Adjacent nodules are frequently aggregated into masses. There are two classes of cases in this group: *1:* The individual may have had simple silicosis at first, followed by the development of asymmetrical tuberculous lesions. This change may occur after many years. *2:* The tuberculous lesions may be manifest first, followed by the silicotic ones in close association. In tuberculosilicosis the roentgenological findings are important. In this condition the hitherto uniformly mottled areas are invaded by one or more asymmetrical areas of shadow with mottling around them. The greatest danger to the workers, aside from the harmful dust itself, lies in the prolonged suspension of the smaller and dangerous-sized particles in the atmosphere. Six important procedures in protection are necessary: (1) To keep the dust particles produced down to a minimum in the atmosphere to be inhaled; (2) to allow the dust to settle as much as possible before it can be inhaled; (3) to remove rapidly the dust-laden atmosphere instead of allowing it to remain stagnant; (4) to separate the particles from the air before they can gain entrance to the respiratory tract; (5) to prevent the spread of tuberculosis; and (6) to appoint satisfactory medical boards for control of the condition.—*A Review of Pneumonoconiosis: further Roentgenological and Pathological Studies, H. K. Pancoast and E. P. Pendergrass, Am. J. Roentgenol. & Rad. Ther., October, 1931, xxvi, 556.*—(*W. I. W.*)

Pneumonoconiosis and Tuberculosis.—The differential diagnosis between pneumonoconiosis and tuberculosis is often difficult. Haematogenously disseminated pulmonary tuberculosis frequently produces roentgen pictures closely resembling diffuse nodular forms of silicosis. The freedom of the apices from changes speaks against tuberculosis and for pneumonoconiosis. Pure forms of pneumonoconiosis were found most in porcelain, cement, and stone-workers. Certain types of pneumonoconiosis tend toward tuberculosis.—*Staublunge und Tuberkulose, W. May, Beitr. z. Klin. d. Tuberk., 1930, lxxiv, 433.*—(*H. J. C.*)

Pneumonoconiosis.—The ore in several underground iron mines in Virginia averages about 39 per cent metallic iron and 35 per cent of silica, with some manganese and phosphorus, and requires drilling and blasting. The authors present the results of a study made of 17 operators of large air drills. These men, exposed to excessive dust, soon developed a moderate cough with slight expectoration, the cough being asthmatic in type, accompanied by wheezing. Conclusions drawn from the study were that, after working four or five years in this excessive dust, these operators may develop distressing symptoms of pulmonary fibrosis after they have discontinued mining and have lived from four to eight years under apparently good physical and hygienic conditions. The fact that such operators at the time of their retirement from years of such exposure have no symptoms may have but little bearing on the future progress of their disease. This form of fibrosis is

not merely a cumulative action of dust, but is a result, a stage of the disease, which requires for its development a certain amount of time after the heavy inhalation of dust. The marked secondary fibrosis is comparatively rapid in its development.—*Pneumoconiosis in Iron Mines, G. B. Lawson, W. P. Jackson and J. E. Gardner, J. Am. M. Ass., April 4, 1931, xcvi, 1129.*—(*G. L. L.*)

Pneumonoconiosis.—Most clinical and statistical studies of silicosis have been made on groups of men still employed in dusty trades. From such statistics one cannot say what is the late effect of short exposures. The authors have reported four cases of silicosis or silicosis and tuberculosis which developed many years after relatively short exposures. These instances suggest the necessity of revising the conception of the length of exposure necessary to produce the disease, and make it seem probable that after relatively short exposures sufficient dust may be deposited in the lungs to set up a progressive fibrosis, which only after many years becomes sufficiently extensive to produce symptoms. They tend to indicate that men in the work develop symptoms only after many years of exposure, not because that length of exposure is necessary but because it takes a long time for the disease to develop.—*Pneumonoconiosis: the Delayed Development of Symptoms, J. A. Britton and J. R. Head, J. Am. M. Ass., June 6, 1931, xcvi, 1938.*—(*G. L. L.*)

Silicosis and Tuberculosis.—The author reports on an experience with 2300 cases. The silicosis developed almost symmetrically in both lungs, but only when the lungs were previously healthy, and especially when there existed no tuberculosis or fibrosis. As to severity, dusty lungs may be divided into two classes, and the more severe stage is frequently complicated by tuberculosis. In these cases marked retraction occurs, with emphysematous areas in the clear spaces. String-like strands to the diaphragm rarely are seen in tuberculosis, but are frequent in silicosis. The tuberculous stage manifests itself with a loss in strength, and especially a loss in weight.—*Über die Entwicklung der Silicosis (Gesteinstaublunge), ihre Beziehung zur Tuberkulose nebst Bemerkungen über ihre Begutachtung an der Hand zon 2300 Fällen, V. Reichmann, Beitr. z. Klin. d. Tuberk., 1930, lxxiv, 452.*—(*H. J. C*)

Asbestosis.—Sections of the lungs of asbestosis cases show fine granular dust, larger fragments of asbestos, and asbestos bodies. The last-named are believed to represent the minute spicules of asbestos fibres which have become thickly covered with protein material. Asbestos appears to act pathogenically in two ways: first, as a direct mechanical irritant and, second, as a chemical irritant. The mechanical-irritant action is suggested in sections by the large amount of fine granular dust, the larger angular particles, and the presence of much fibrous tissue with multinucleated foreign-body giant cells alone. The chemical-irritant action is believed to be due to the slight solubility of asbestos. This leads to the formation of the asbestos bodies and induces pulmonary fibrosis.—*Asbestos Dust and Asbestosis Bodies from the Lungs of an Asbestos Worker, W. E. Cook, J. State Med., September, 1931, xxxix, 544.*—(*J. P. B.*)

Asbestos-Like Bodies in Lungs.—A man 71 years of age was admitted to the hospital following several attacks of haemoptysis, with subsequent fever and delirium. Although the physical signs and X-ray findings were not typical, tubercle bacilli were demonstrated in the sputum. At autopsy the general appearance of the lungs suggested chronic fibroid tuberculosis, and anthracosis was quite marked. In the upper parts of the upper lobes there was a patchy overgrowth of fibrous tissue, which here and there contained brownish bodies singly or in groups of five to ten. They were slender rods with bulging ends, and on microscopic examination showed a black central core with a semitransparent brown outer lamina. The segmentation which occurs in asbestos bodies was absent. On treatment with potassium ferrocyanide and hydrochloric acid the brown material took on an intense blue color, showing the presence of free iron. These bodies, although similar to asbestos bodies in size, shape, color, and their content of iron, differed from them in being coarser, less regular, unsegmented, and in possessing a black opaque core. They did not cause a very serious fibrous reaction. It is of note that the man had worked only in Alabama and Lancashire mines, which are largely coal or iron, and had had no contact with asbestos mining.—*A Case of Asbestos-Like Bodies, F. E. Tylecote and J. S. Dunn, Lancet, September 19, 1931, ccxxi, 632.*—(*R. D.*)

Pulmonary Asbestosis and Tuberculosis.—The subjects of certain forms of pneumonoconiosis are prone to develop pulmonary tuberculosis. The discovery that asbestos workers are often the victims of occupational disease raises the question whether the incidence of tuberculosis in the group is increased. Merewether found active tuberculosis in 3 of a total of 374

working asbestos operatives. The group reported here includes, however, those who had changed their occupation and is limited to those who came to a clinic because of chest symptoms. Among 57 cases, 18 were men and 39 were women; one of the men and 9 of the women had active tuberculosis. These active cases were classified as follows: generalized tuberculosis as a terminal event in one; acute phthisis in two; extensive subclavicular infiltration in two; chronic fibroid phthisis in five. Two other cases showed obsolescent disease at autopsy. The symptoms of pulmonary asbestosis are irritable cough, scanty expectoration, shortness of breath progressing to a distressing dyspnoea on the slightest exertion, pains in the chest, loss of weight, "earthy" complexion, and slight clubbing. Physical signs include impaired resonance at the bases, and fine dry crackles over the bases and in the axillae. Sputum examination reveals asbestos bodies. The X-ray shows a diffuse fine fibrosis affecting chiefly the lung bases and sparing the apices and infraclavicular regions. Four cases came to autopsy, two showing obsolescent tuberculosis (in the mediastinal lymph nodes in one, in the abdominal lymph nodes in the other), which had remained quiescent despite 8 and 9 years of exposure to asbestos dust. The other two cases showed disease of the caseous type with little development of fibrous tissue. Asbestos fibres were present in the caseous areas as well as asbestos bodies. It is impossible to draw definite conclusions from this small record as to whether tuberculosis or asbestosis was the antecedent disease. In two cases the exposure to asbestos dust preceded the onset of signs of tuberculosis by 6 and 15 months; in the others the exposure preceded the signs of tuberculosis by from 4 to 13 years.—*Pulmonary Asbestosis Complicated by Pulmonary Tuberculosis, W. B. Wood and S. R. Gloyne, Lancet, October 31, 1931, ccxxi, 954.*—(*R. D.*)

Actinomycosis of Lungs.—Pulmonary actinomycosis is difficult to diagnose clinically. It resembles chronic empyema and tuberculosis especially, but it frequently presents multiple fistulae and hard infiltration of the thoracic wall. In a patient with these latter signs and with pulmonary symptoms, this disease should be suspected and the sputum examined repeatedly. In the film there is often destruction of the ribs or sternum or periosteal thickening. Pleural involvement is nearly always present. Study of 14 cases revealed the fact that in eight an abdominal abscess preceded the pulmonary symptoms. The signs were wholly pleural in 4 cases and wholly pulmonary in one. The most important signs in the diagnosis are (1) roentgenological signs of destruction of bone or of periosteal thickening; (2) induration of the costal wall; (3) presence of the fungus in the sputum or discharges; and (4) history of previous abdominal abscess. Potassium iodide and irradiation offer a great deal if given early in the disease.—*The Roentgenologic Aspects of Actinomycosis of the Lungs, B. R. Kirklin and H. W. Hefke, Am. J. Surg., July, 1931, n. s. xiii, 1.*—(*H. S. W.*)

Bronchomoniliasis.—This, the most common manifestation of monilia infection, occurs chiefly in tropical and subtropical climates. Most cases reported have been in adults. The mode of infection is undetermined. Moniliae are saprophytic in vegetable debris and in the human mouth. They grow best on media containing sugar. They may be divided, as regards pathogenicity, into 3 groups, as follows: *1: Nonpathogenic:* Inoculation of rabbits' lungs produces neither pulmonary lesions nor general infection. *2: Virulent,* but secondary invaders: Injection into rabbits' veins and lungs causes fatal septicaemia without localized nodular pulmonary lesions. *3: Those causing Bronchomoniliasis:* Injection into rabbits' lungs produces characteristic pulmonary lesions. Primary bronchomoniliasis may simulate pulmonary tuberculosis, chronic bronchitis, bronchial asthma, or pneumonia. Myocarditis and decompensation of the right side of the heart occur in some cases. Usually, there is a yeast-like odor to the breath. The mouth should be washed with potassium-permanganate solution before the sputum is collected. Castellani's criterion for a positive diagnosis is that injection of the material into the lungs of a rabbit must produce the characteristic pulmonary lesions. Balog and Grossi used as an antigen emulsions of untreated *Monilia pinoyi Castellani* intracutaneously and obtained positive reactions in 18 patients, with bronchomoniliasis, and negative reactions in 53 controls, which consisted of normal persons or those having tuberculosis or other disease not due to moniliae. An antigen made from killed moniliae gave a nonspecific reaction. Tuberculosis and bronchomoniliasis may be concurrent. The apices of the lungs usually are clear in uncomplicated bronchomoniliasis. To determine whether tubercle bacilli are present in the sputum, guinea-pig inoculation should be done. If present, they are assumed to represent the primary invaders. Cases of uncomplicated tuberculosis should be isolated from cases of moniliasis. In cases of concurrent moniliasis and tuberculosis the moniliasis should be treated first.

A case is reported to illustrate primary bronchomoniliasis complicated by pulmonary tuberculosis. The tubercle bacilli were considered the secondary invaders. In secondary moniliasis the mucous membranes of the mouth, pharynx and bronchi usually are invaded. Such invasion is rare in the primary type. In addition to a symbiotic action between moniliae and tubercle bacilli other cases of mixed infection of the lungs have been reported. Secondary bronchomoniliasis is not infrequently found in cases of cancer and diabetes. A case is reported illustrating concurrent bronchomoniliasis and syphilis. The presence of syphilis of the lungs was not proved in this case. Bronchomoniliasis is curable with iodides if treated early. Other forms of treatment have met with varying success. Necropsies have been mentioned by only 5 authors. A summary of the author's case of primary bronchomoniliasis follows: A woman, aged 38, had influenza in 1918, typhoid fever in 1919, and pleurisy with effusion on the left in 1920. The fluid was not examined bacteriologically. The pleurisy recurred in 1921 and pulmonary tuberculosis was diagnosed though the sputum was negative for tubercle bacilli. In 1923 cough, blood-streaked sputum, hoarseness, easy fatigue, fever and dyspnoea developed. No weight was lost. Physical examination six months later revealed moist râles and signs of thickened pleura over the lower half of the left lung. Fibroid tuberculosis was diagnosed. Repeated sputum examinations throughout the illness were negative for tubercle bacilli. Cultural methods were not used. A roentgenogram revealed a homogeneous density of the lower half of the left lung with displacement of the mediastinum to that side. Two small areas of similar density were present in the middle third of the right lung. Eight months were spent in a sanatorium, where therapeutic tuberculin was given. Clinical improvement occurred, though the densities in both lungs extended. Acute febrile illness developed, with pain in the left hemithorax, cough and blood-streaked sputum. The pulmonary lesions became still more extensive. Thoracentesis secured no fluid. Blood counts were normal. A pure growth of *Monilia pulmonalis Castellani* was obtained from the sputum. Iodides were given in large doses, orally and intravenously. The only apparent effect was increased expectoration. Death occurred in December, 1924, with dyspnoea, cyanosis, and subcutaneous oedema. There was never definite roentgenographic evidence of pulmonary excavation. The necropsy findings were as follows: The left lung was one-third normal size and almost airless. The pleurae were thickened and adherent. Small cavities were uniformly distributed throughout. The largest was 4 cm. in diameter. The lymph nodes were small, pigmented black, and only one contained lime salts. The right lung was increased in size, pale and mottled. The pleurae were slightly thickened and adherent. Approximately 85 per cent of the lung was consolidated. A few cavities were present. The cut-surface resembled that of colloid carcinoma. The pericardium was drawn to the left by adhesions. There was passive congestion of the liver. The spleen contained several tubercle-like bodies. Microscopical findings in the lungs and lymph nodes are described in detail. There was no evidence of tuberculosis.—*Bronchomoniliasis: A Clinical and Pathological Study with Report of Illustrative Cases, O. S. Warr, Ann. Int. Med., September, 1931, v, 307.*—(*R. M. F.*)

Relation of Echinococcus Infection to Pulmonary Tuberculosis.—The author reports a case in which two years after the healing of an echinococcus cyst there resulted a disseminated tuberculosis of both lungs, worse, however, in the echinococcus lung. The differential diagnostic points between the two diseases are discussed.—*Lungenechinokokkus und seine Beziehung zur Lungentuberkulose, H. R. Gebrandy, Ztschr. f. Tuberk., 1930, lv, 451.*—(*H. J. C.*)

Yeast Infection of Lung.—Five cases of yeast infection of the lungs are reported. The symptoms varied from signs of slight bronchitis with mucopurulent expectoration to hectic fever and haemorrhagic expectoration. The diagnosis was based on the absence of the tubercle bacillus and the constant presence of yeast cells in the sputum. Yeast-mold infection of man is rare, and usually occurs as skin infections. In rare instances it does not remain localized to the skin but gains entrance into the body and produces tubercle-formation, tissue destruction and abscess-formation in the lungs, spleen and kidneys. The fungi may be absorbed in the lymph-stream after the skin is invaded, and secondary infection may occur in distant organs, while the pulmonary type of infection may occur from inhalation of dust containing the mold. The disease affects all ages, but is found most often after middle life. Blastomycosis is practically confined to Illinois and is found in and about Chicago.—*Yeast Infection of the Lungs, T. R. Healy and L. B. Morrison, Am. J. Roentgenol. & Rad. Ther., September, 1931, xxvi, 408.*—(*W. I. W.*)

Cancer of Upper Air-Passages.—The British Empire Cancer Campaign, the National Radium Commission, and the Medical Research Council have now 19 centres with adequate facilities for radiotherapy in the treatment of malignant tumors. Early diagnosis is important and the choice of treatment is difficult. Clinically and experimentally, it has been noted that the best results obtained with radium occurred when it was applied in close apposition to the lesion. It is important to irradiate uniformly the whole of the diseased area. This is done more certainly with needles than with seeds. The use of small doses over long periods has given better results than large doses applied for a short time. Syphilitics, chronic alcoholics and persons seriously debilitated rarely respond well to radiation. Postirradiation changes may appear as total disappearance of growth, fibrosis, oedema, radiation sores, necrosis, and pain. Intrinsic carcinoma of the larynx can be diagnosed early while it is an entirely local disease, and surgery gives good results. Extrinsic growths starting in the skin of the nose are either rodent ulcer or squamous-cell carcinoma. In all types of rodent ulcer, radiation is better than excision, but in resistant growths excision by diathermy is indicated and preferable to the knife. In the nasal sinuses there is a great variation in the types of growth, but a cylindrical carcinoma of the adenocarcinomatous type is most common. They tend to recur locally, but glandular involvement is late. Surgery combined with diathermy and radiation have given improved results. In sarcoma of the nasal sinuses, X-ray and surface radium treatment should be tried before surgery. Carcinoma is the most common tumor of the nasopharynx. Radiation should be applied before confirmation of the diagnosis by biopsy. The same is true of carcinoma of the tonsil and mesopharynx. The majority of growths in the palate and cheek are squamous-cell carcinoma and should be given a preoperative dose of X-ray and radium. In the author's clinic, out of 400 cases, 19 per cent of cases of carcinoma, 50 per cent of cases of sarcoma, and 47 per cent of cases of endothelioma are surviving. He believes that there is a great future for radiotherapy, if the treatment is properly handled, but it is necessary to have special clinics, richly endowed, vigorously supported by those in control of radium, and staffed by groups of specialists.—*Radiotherapy in the Treatment of Cancers of the Upper Air Passages. Semon Lecture by W. Douglas Harmer, Brit. M. J., November 14, 1931, no. 3697, 910.*—(*M. E. P.*)

Cancer of Lung.—A woman of 49 complained of pain and swelling of the left leg which had been present since a trivial accident four months previously. She had been bedfast from the day after the injury, since which time she had had an unproductive cough. She had had haemorrhoids for four years. Her physical examination revealed pigmented naevi on the right cheek; small, firm lymph nodes about the left clavicle; retraction of the upper medial part of the left chest where there were diminished tactile fremitus and dulness and in the corresponding region of the back. The breath-sounds were amphoric in this region. Varicose veins were conspicuous, especially in the left leg, and there were huge haemorrhoids. A diagnosis was made of pulmonary neoplasm with metastases to the lymphoid tissue. The X-ray revealed "massive increase in density adjacent to the heart extending upward and medially to obscure the space between the first and third ribs (anterior projection)." The patient lived for 13 days after admission. Her temperature ranged to 101.2°. She received palliative deep X-ray therapy. Autopsy revealed "carcinoma of the left upper bronchus, with widespread metastasis; cardiac thrombosis; pulmonary thrombosis and infarction; hydrohemopericardium; acute splenic tumor; renal infarction; surgical skin wounds; multiple papillomas of skin; lineae albicantes; obesity; chronic cholecystitis; cystic changes in ovaries; varicose retroperitoneal veins." The tumor was an adenocarcinoma with, however, areas of distinct squamous-cell type of cancer. The sudden death of the patient was probably due to the haemorrhagic infarction of the lung and not to the X-ray therapy, the dose of which was small.—*Adenocarcinoma of the Bronchus, with Widespread Metastases; Case Report, W. S. Middleton, E. A. Pohle and G. Ritchie, Radiology, June, 1931, xvi, 945.*—(*H. S. W.*)

Cancer of Lung.—Morgagni (1682–1772) was the first to publish the results of several autopsies on lungs that might be diagnosed as cancerous and were so reported by him. Of recent years tumors of the lung have been more frequently found, and there is evidence that the incidence of primary carcinoma of the lung is increasing. Among 81 patients at Kings County Hospital who presented evidence of malignant tumors in the lungs there were 8 with primary carcinoma proved at autopsy, 35 believed to be carcinoma on the basis of the history, physical examination, X-ray and bronchoscopic findings, 20 with metastatic carcinoma in the lung, 10 with metastatic lymphosarcoma, 6 with

metastatic sarcoma, 1 with metastatic hypernephroma, and 1 with metastatic teratoma. The commonest symptoms were cough, with or without expectoration (which if present was usually bloody at some period of the disease), dyspnoea, pain, cyanosis, weakness, and emaciation. Little benefit is to be anticipated from the treatment of secondary deposits. Early diagnosis of the primary lesions may permit treatment and is therefore desirable. To secure such diagnosis attention should be paid to persistent cough without demonstrable cause, haemoptysis in the absence of tubercle bacilli, weakness, and loss of weight which does not respond to treatment. The primary pulmonary carcinoma may arise from (1) the bronchial epithelium, in which case it causes bronchial obstruction and bronchiectatic cavities, (2) the mucous glands of the large bronchi, in which case invasion of the wall and obstruction may occur, and (3) the alveolar epithelium, in which group infiltrating masses which spread rapidly may be observed. Attention is called to the fact that a pleurisy with effusion in a person of middle age or later should suggest the question of the presence of pulmonary or mediastinal new growth. Such an effusion, if bloody, is almost pathognomonic. It is also to be noted that the presence of tubercle bacilli in the sputum does not exclude neoplasm. Eleven case histories are cited, illustrating the symptomatology and methods of diagnosis.—*Malignancy in the Lung: Including Eight Primary Carcinomas with Autopsy Findings, H. M. Moses, Ann. Int. Med., December, 1931, v, 765.*—(*R. D.*)

Cancer of Lung.—The pathognomonic signs in 6 cases of primary carcinoma of the lungs are reported. An analysis of these cases reveals that pain occurred in 4, effusion in 2, asthmatic symptoms in 2, and haemoptysis in 4. In all of these cases the heart was drawn over to the affected side during inspiration and away from it during expiration.—*Primary Carcinoma of the Lungs, J. Polevski, Arch. Int. Med., December, 1931, xlviii, 1126.*—(*W. I. W.*)

Difficult Diagnosis of Cancer of Lung.—A case of pulmonary disease, requiring careful differential diagnosis, presented a sensation of intrathoracic pressure, haemoptysis, fainting and an X-ray shadow the size of an apple in the upper pulmonary field. It led to a consideration of aortic aneurysm, mediastinal and pulmonary tumor, pulmonary abscess and echinococcus cyst. An oblique film excluded aortic aneurysm and mediastinal tumor. Abscess was ruled out on the strength of the symptoms. Echinococcus cysts usually lie in the midlung field and mostly in the lower lobe. The complement-fixation test for echinococcus was positive; the skin reaction with echinococcus antigen was negative. The patient rapidly grew worse with hoarseness, sharp dyspnoea, dysphagia, and fever, and died following an operation, in which 3000 cc. of blood were removed from the thoracic cavity. Postoperative diagnosis was carcinoma of the lung.—*Lungenechinokokkus oder Lungentumor? G. Lepehne, Deutsche med. Wchnschr., July 3, 1931, lvii, 1156.*—(*H. S. W.*)

Cancer and Tuberculosis.—In the course of the experimental work on the relation of cancer to tuberculosis the gastrosplenic region was examined by microscopic section in 625 mice. About one-half of these had been inoculated with tubercle bacilli; the others were control animals of the same strain, kept under similar conditions. In the inoculated animals a small-cell infiltration, consisting almost exclusively of lymphocytes, is often found in the fat about the splenic vein as well as in other parts of the body. Acid-fast bacilli are not common, but they have been found in the spleen and enlarged lymph nodes and among small dense collections of lymphocytes. In the second year of these experiments an outbreak of pseudotuberculosis began which involved both inoculated and control mice. The histological conditions, involving the pancreas in 60 mice dying of pseudotuberculosis, were compared with those found in 60 mice which had been inoculated with tubercle bacilli and were free from pseudotuberculosis. In the former group there was an exudation of cells, chiefly of the polymorphonuclear type, which may run on to the formation of adhesions to the adjoining organs. No sign of proliferation of the secreting cells of the pancreas was found. In the second group the infiltration of the pancreatic area was mainly lymphocytic in character. Proliferation of the secreting cells of the pancreas and signs of activity in the fibroblasts of the adhesions became common. In the 60 mice there were 5 instances of carcinoma of the pancreas, a condition not hitherto described in this animal. The action of many agents which may form the basis of a malignant growth is regularly accompanied by a lymphocytic reaction. In a former paper the author pointed out that the lymphocytic reaction appears to be the connecting link between the tubercle bacillus and malignant tumor. Ewing remarked that the process excited by the tubercle bacillus may "progress of its own momentum and gradually acquire

neoplastic properties." This momentum is proportional to the crowding of the lymphocytes in places where anaerobiasis is easy. The acquisition of neoplastic properties depends on the supply of growth-stimulating substances coming from an anaerobic mass of cells. Ninety years ago Walshe stated that "tubercle and cancer possess a sort of repulsion in respect of each other." He found only 7 cases of active phthisis in 104 autopsies of cancer. In 1929 Pearl published the evidence obtained from the records of the Johns Hopkins Hospital. He confirmed the findings of the older pathologists, establishing the fact that only 7 per cent of those dying of cancer had active tubercle, and conversely that those dying of phthisis very seldom had tumor. In 1924, from an analysis of the deaths in the occupations at three census periods, the author showed that the incidence of cancer in nearly all large occupations in 1911 was proportional to the incidence of phthisis in the same occupations in 1891, and that the increase in deaths from cancer cannot be explained by the increase in the number of men who attain the age of 55. Later corresponding results were shown to hold for the whole adult population of England and Wales. The combined deaths from phthisis and cancer were always close to one-fifth of the total deaths after the age of 25. The increase in cancer regularly compensated for the decline in phthisis. There is good evidence that the great majority of the adult population has been in contact with tubercle bacilli and that opportunities for reinfection are still very common. Hence the following questions require to be answered in the light of experimental work: *1:* Is it possible that tuberculosis and most cancers are manifestations of the same essential condition, the resistance of the body to invasion by the bacillus? *2:* May not successful resistance to the bacillus in early life prepare the body to deal with a second invasion by different methods? *3:* If the acquired resistance to tuberculosis is the usual predisposing cause of cancer, may not the second invasion be the factor which precipitates the crisis in the life-history of the cells of the body?—*Cancer and Tuberculosis: the Development of Malignancy, T. Cherry, Lancet, February 7, 1931, ccxx, 285.*—(*F. G. P.*)

Malignant Haemangioma of Lung.—This case of a child 4 months and 20 days old was the first instance of primary neoplasm of the lung on record at the Babies' Hospital in a series of over 5,900 autopsies. There had been 4 cases with metastases from embryonal adenosarcoma of the kidney. When about two months old the child had had attacks of wheezing respiration associated with cyanosis. When 4 months old a bronchoscopy was performed, and distortion at the mouths of both main bronchi, more marked on the left, was noted. An X-ray showed displacement of the trachea to the left and a dense, even shadow of the right upper lung field. At autopsy the posterior third of the right upper lobe was found to be deep red and fleshy in consistence, but not firm. The anatomical diagnosis was malignant haemangioma of the right lung, bronchi, small intestine, pancreas, suprarenal gland, adipose tissue, abdominal sympathetic ganglion, scalp, and lip. The literature contains reports of malignant haemangioma, but in no other instance was the principal and largest mass located in pulmonary tissue.—*Malignant Hemangioma of the Lung with Multiple Visceral Foci, M. Wollstein, Arch. Path., October, 1931, xii, 562.*—(*R. D.*)

Pleural Fluids Complicating Intrathoracic Tumor.—In 131 autopsies on intrathoracic malignant disease at Victoria Park between the years 1911 and 1930, fluid was found in 56 instances. Thirty-eight were clear effusions and 18 were purulent. Of the 38 serous fluids 10 occurred with secondary deposits in the pleura, and probably resulted from these deposits. Four appeared to result from underlying sepsis (three bronchiectasis and one gangrene of the lung). The remainder may possibly be accounted for either by minute subpleural deposits of growth not visible to the naked eye, or by pressure in the mediastinum causing interference with lymphatic drainage or venous return. Two serous effusions had .contained tubercle bacilli during life. Pulmonary tuberculosis and intrathoracic new growth occurring in the same lung are not uncommon. Three instances occurred of serous effusions which became absorbed with advancing new growth occluding the pleural sac. Of the 18 purulent fluids 5 consisted of pus from bronchiectatic cavities, 2 were instances of pyopneumothorax, and 11 were empyemata (five resulting from underlying sepsis in the lung, and one following subphrenic abscess). The danger of confusing a bronchiectatic cavity with empyema in these cases is evidently considerable.—*Pleural Fluids Complicating Intrathoracic New Growths, S. R. Gloyne, Lancet, February 7, 1931, ccxx, 294.*—(*F. G. P.*)

Nature of Hodgkin's Disease.—The nature of Hodgkin's disease is still very uncertain. One school maintains that the disease is neoplastic in nature. The ad-

herents of this view maintain that the tumor is primary in the lymphoid tissues of the body. A second school believes that Hodgkin's disease is of an infectious nature. Bacteriological studies by different investigators have led to no single microörganism as the aetiological factor. The tubercle bacillus has been held to be the inciting agent by some, because tubercle bacilli have been obtained from Hodgkin's tissues. The irregularity of these findings has led to a belief that to obtain Hodgkin's lesions it is necessary to have a certain type of tubercle bacillus which has a certain degree of pathogenicity. Recently L'Esperance has presented evidence which suggests that the avian tubercle bacillus might be the aetiological factor. In the study of tuberculous lesions in experimental animals the writer noted the occurrence of numerous giant cells in lung, liver, spleen and lymph nodes, which he was unable to differentiate from the giant cells seen in Hodgkin's lesions. It was concluded that these cells which resemble the Sternberg giant cells of Hodgkin's disease were megakaryocytes. At certain stages of acute tuberculosis there was found a marked hyperplasia of the megakaryocytes in the marrow, with a wandering out of these cells into the circulation and thence into the various tissues involved in the tuberculous process. This phenomenon was not observed in chronic tuberculosis, although it is possible that it exists to a slight degree. The above facts led to a study of the histopathology of Hodgkin's disease, with the possibility in view that the megakaryocyte might be the cell type primarily involved in the pathological process. The material studied consisted of 22 autopsies and about 100 surgical specimens. The number of bone-marrow specimens was very few. A biopsy specimen from the skin of an individual suffering from myelogenous leukaemia was also studied. This individual had numerous nodules in his skin in various parts of the body. In determining the possible relation of Hodgkin's disease to the megakaryocyte interest was chiefly centered on the bone-marrow. The complex picture of Hodgkin's disease can be demonstrated in a single section of bone-marrow. The points to be emphasized are (1) hyperplasia of the marrow with a marked increase of immature cells which probably are the progenitors of megakaryocytes, and (2) presumptive evidence that the giant cells (megakaryocytes) are the end-results of fusion of several premegakaryocytes. The Hodgkin's lesions, as seen in the various tissues of the body, differ in no essential respect from those in the bone-marrow. A new interpretation of the disease is here presented. It is suggested that Hodgkin's disease is a malignant process of the bone-marrow. This conception is based on the probability that there is a single parent cell for haematopoiesis in the marrow. The presence of nucleated red blood cells and of myelocytes in the blood-stream in Hodgkin's disease, and of abnormal platelets and of myelocytes in cases of grave erythroblastic derangements supports the theory. The parent cell must be multipotential. From this stem arises the erythrocyte, the megakaryocyte and blood platelets, the basophile, eosinophile and neutrophile. If Hodgkin's disease is one in which the megakaryocyte is the cell type chiefly involved, then it becomes apparent that it is closely related genetically to the myeloid leukaemias and to the erythroblastic dyscrasias. Many observers have noted the marked increase of blood platelets and the presence of enormous blood platelets in the circulating blood in Hodgkin's disease. Since Wright has so clearly demonstrated that megakaryocytes are the cells from which platelets arise, the findings relative to the platelets in Hodgkin's disease fit in readily with the notion that this disease is primarily an involvement of the megakaryocyte. Another common occurrence in Hodgkin's lesions is fibrosis. This occurs in bone-marrow as well as in the lymph nodes and other tissues. This may be explained as follows: If the tumor cell is the megakaryocyte, then there will be a large production of blood platelets in the tumor nodules. Blood platelets are closely connected with the formation of fibrin. Wherever there is much fibrin formation, there is an organization of the fibrin by fibroblastic proliferation. This would lead to the marked fibrosis commonly seen in the more chronic Hodgkin's lesions. By placing the primary lesion in the bone-marrow one can more easily understand the irregular distribution of the lesions in the body. Lesions outside of the marrow may be considered metastatic tumor growths. These metastases probably take place through the blood-stream. Why the tumor finds lymphoid tissue especially suitable for metastatic growth is an unsolved question. From the above discussion it would seem as though Hodgkin's disease should be removed from the group of infectious diseases and placed as a malignant process of the haematopoietic tissues of the bone-marrow. This also would imply the removal of this disease as a primary neoplasm of the lymphoid tissue. The involvement of tissues outside of the bone-marrow could be logically regarded as metastatic growths. A certain amount of evidence is presented which suggests that the cell primarily involved in the

pathology of Hodgkin's disease is the megakaryocyte. To denote that the predominant phase of Hodgkin's disease, which distinguishes it from other bone-marrow dyscrasias, is the primary involvement of the megakaryocyte, the term "megakaryoblastoma" is suggested to designate real Hodgkin's disease.—*An Interpretation of the Nature of Hodgkin's Disease, E. M. Medlar, Am. J. Path., September, 1931, vii, 499.—(J. S. W.)*

Tuberculous Lesions Resembling Hodgkin's Disease.—Numerous investigators have reported the association of tuberculosis and Hodgkin's disease. Ewing states that "tuberculosis follows Hodgkin's disease like a shadow." L'Esperance is of the opinion that the tubercle bacilli which she obtained from certain cases of Pel-Ebstein disease were of the avian type, and believes that avian tubercle bacilli have an aetiological relationship to Hodgkin's disease. In experimental animals, however, no one has been able to produce the gross and histological pathological changes of Hodgkin's disease with tubercle bacilli. The present study has to do with the interpretation of certain lesions resembling Hodgkin's disease in experimental tuberculosis. Rabbits infected intravenously did not give the gross changes of Hodgkin's disease. Microscopic examination of the various tissues, however, showed a few to many giant cells, which were indistinguishable from the giant cells seen in Hodgkin's disease. From a study of the bone-marrow in these animals it was concluded that the giant cells observed in the tissues were megakaryocytes. In rabbits receiving a small dose of avian bacilli or partially protected by vaccination, no lesions suggestive of Hodgkin's disease were observed in the tissues. Apparently, only in acute avian tuberculosis did the megakaryocytes wander out of the marrow in sufficient numbers to enable their detection in the tissues. Rabbits, guinea pigs and calves, extensively infected with virulent bovine tubercle bacilli, showed numerous groups of megakaryocytes in lymph nodes and the spleen. Guinea pigs infected with virulent human bacilli gave similar results. Lesions resembling Hodgkin's disease were not observed in any of these animals not heavily infected or living a long time with a chronic tuberculosis. Recently, the same type of lesion was observed in the lung tissue of a human being who died from an extensive acute "spread" of his tuberculosis. Studies of the lung sections in this case showed numerous megakaryocytes in the part of the lung involved in the recent "spread," whereas in the more chronic lesions such cells were not observed. Here again we have the presence of megakaryocytes in the acute tuberculous process and not in the chronic. *Summary:* Lesions resembling Hodgkin's disease are often present in acute tuberculous foci. These lesions are simply the accumulation of megakaryocytes which have emigrated from the bone-marrow to the areas of damaged tissues. They are not true Hodgkin's lesions. The participation of the megakaryocyte in the disease process is not specific for tuberculosis. It is necessary to exercise great care in diagnosing Hodgkin's disease from tissue sections. The presence of the Sternberg type of giant cell in tissue is not sufficient evidence to warrant a diagnosis of Hodgkin's disease. Various types of virulent tubercle bacilli cause the production of the lesions which simulate Hodgkin's disease. This would seem to preclude the possibility that any one type of tubercle bacillus is the aetiological agent of Hodgkin's disease. Lesions suggestive of Hodgkin's disease have been observed only in acute tuberculosis. In chronic tuberculosis these lesions have not been encountered.—*The Significance of Lesions Resembling Hodgkin's Disease in Tuberculosis, E. M. Medlar and K. T. Sasano, Am. J. Path., September, 1931, vii, 491.—(J. S. W.)*

Lymphoblastoma.—The roentgenological manifestations in the mediastinum and lungs are described in 40 cases of Hodgkin's disease, 17 of lymphosarcoma and 10 of leukaemia. Among the 40 patients with Hodgkin's disease, 26 were males and 14 were females, the average age of whom was thirty-two years. Seventy-five per cent of all patients were between the ages of twenty and forty years; the oldest was fifty-five and the youngest seven years. In the group with lymphosarcoma the average age was thirty-eight years; the oldest being fifty-five and the youngest six years. There were 10 males and 7 females. In a small group of patients with leukaemia, the average age was thirty-seven years; the oldest was aged forty-eight years and the youngest eight years. Six were males and 4 females. Routine roentgenological examination revealed evidence of thoracic involvement in about 49 per cent of the cases in which the diagnosis was Hodgkin's disease of any part of the body and in about 20 per cent of the cases of lymphosarcoma and leukaemia. The roentgenological signs of Hodgkin's disease in this series of cases were classified into two main groups. The first group comprised those cases in which the lymph nodes only were involved, either those of the mediastinum only, or the more or less separated hilar nodes, or both. In the

second group were the cases with involvement of the pulmonary tissue, either infiltrative or metastatic, with or without mediastinal lymph-node involvement. Of the 40 cases, 62.5 per cent belonged to group 1. Twenty-five of them presented involvement of the nodes of the mediastinum and hilum, and the lungs were free from disease. In 5 cases definitely localized lymph nodes were demonstrable in one hilum or in both. In the 40 cases localized lymph nodes were present on one or both sides of 32.5 per cent, or 13 cases. Invasion of the lungs was seen in 12 cases, or 30 per cent. The 17 cases in which lymphosarcoma was microscopically confirmed were grouped in the same manner. Localized, discrete hilar shadows were observed in 4 cases. These sarcomatous changes in the lung were of the infiltrative type in 2 cases and originated in the lymph nodes. The disease was unilateral in 5 cases. The phenomena in the 10 cases of leukaemia with mediastinal or pulmonary involvement were little different. In all cases there were definitely enlarged mediastinal or hilar nodes; the nodes were discrete in 5 cases. The differential diagnosis must take into account aneurysm, beginning tumors, substernal goitre, carcinomatous or sarcomatous metastasis, primary carcinoma of the lung or bronchus, tuberculosis of the lungs, Pott's abscess of the thoracic portion of the spinal column, marked dilatation of the oesophagus, and mediastinitis.—*Roentgenologic Study of Intrathoracic Lymphoblastoma, B. R. Kirklin and H. W. Hefke, Am. J. Roentgenol. & Rad. Ther., November, 1931, xxvi, 681.*—(*W. I. W.*)

Amyloid of Spleen.—Follicular amyloid is found most frequently in tuberculosis in youthful individuals, while amyloid of the pulp is noted in syphilis and in older patients. In sago spleens the nodules are large and contain small blood-vessels while in lardaceous spleens they are small and free from vessels. As a whole it is not the age nor the disease so much as the vascularization and function of the lymphatic apparatus that determines the site of the amyloid. In youth the follicles are vascular and actively functioning, while at an older age the opposite condition obtains. The predominance of sago spleen in tuberculosis and of the lardaceous spleen in syphilis is due to the frequency of tuberculosis in youth and that of tertiary syphilis in later ages. Otherwise the course of the basic disease is similar. Mild follicular disease can produce amyloid of the pulp in youth and follicle irritation can produce sago spleen in the aged.—*Die Beziehung der Amyloidablagerung zur Vascularisation in der Milz, K. Higuchi, Virchow's Arch., 1930, cclxxix, 538.*—(*H. J. C.*)

Gastric Ulcer and Pulmonary Tuberculosis.—Figures obtained from a study of 7,700 necropsies at Bellevue Hospital showed the incidence of gastric ulcer and scars to be 120, and of duodenal ulcers and scars 44. There were 86 open gastric ulcers and 35 open duodenal ulcers. Deducting for open duodenal and gastric ulcers in the same person, there were 117 cases of peptic ulcer. Among the 7,700 necropsies studied, 891 patients were recorded as having active pulmonary tuberculosis. Among the 120 patients with gastric ulcers and scars there were 43 who had suffered from active pulmonary tuberculosis at the time of death. Of 44 patients showing at necropsy either healed or open duodenal ulcers, 10 had active tuberculosis. Further striking association is shown in other groups of pulmonary and pleural disease. Pneumonia, bronchitis and emphysema were frequently associated with peptic ulcer.—*Peptic Ulcer: Association with Pulmonary Tuberculosis, M. Sturtevant and L. L. Shapiro, Arch. Int. Med., December, 1931, xlviii, 1198.*—(*W. I. W.*)

Bacteriology and Serology of Rheumatism and Tuberculosis.—Following the recent publications of Löwenstein and his coworkers, attempts were made to isolate tubercle bacilli from the blood of tuberculous patients and of patients with acute polyarthritic rheumatism. In 85 cases of active tuberculosis and in 9 cases of acute rheumatic fever all culture tubes, more than 350, remained sterile. Since these bacteriological studies remained unsuccessful serological studies were undertaken. The reaction according to Lehmann-Facius was used. A total of more than 600 patients were examined. The reaction was positive in a large percentage of tuberculous patients. In more than 200 patients in which tuberculosis was excluded with certainty the reaction was negative. Having thus established a satisfactory sensitivity and a high-grade specificity of the reaction, patients with acute and subacute polyarthritis were tested. Of 23 such patients 16 gave a positive reaction. It is concluded that in many cases of rheumatic polyarthritis tuberculosis plays an important rôle which is, however, not necessarily the aetiological rôle, strictly speaking.—*Bakteriologische und serologische Untersuchungen bei Tuberkulose und "rheumatischen" Erkrankungen, H. Horster, Klin. Wchnschr., December 26, 1931, x, 2389.*—(*M. P.*)

Insulin in Diabetes with Pulmonary Tuberculosis.—The author warns against the use of too large doses and too great an increase in weight in treating diabetes with insulin. In spite of a definite improvement in metabolism a number of the cases showed a progression of the tuberculosis.—*Insulinbehandlung bei Diabetes mit Lungentuberkulose, H. Weskott, Beitr. z. Klin. d. Tuberk., 1930, lxxiv, 89.*—(*H. J. C.*)

Tuberculosis and Diabetes Insipidus.—Because of an unusual case of combined pulmonary tuberculosis and diabetes insipidus, the authors discuss the relation between pulmonary tuberculosis and the constitution or the vegetative system.—*Tuberkulose und vegetatives System, D. Epstein and P. Tarkanowski, Ztschr. f. Tuberk., 1930, lvii, 167.*—(*H. J. C.*)

Tuberculosis and Pregnancy.—The prognosis in women in whom tuberculosis was first discerned during pregnancy is less favorable than in cases in which tuberculosis has existed for some time. Collapse therapy in suitable cases is a valuable aid in preventing progression of the tuberculosis.—*Über Lungentuberkulose bei Schwangerschaft, K. Lydtin and R. Linde, Ztschr. f. Tuberk., 1930, lvi, 329.*—(*H. J. C.*)

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